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Editorial
Mercury Toxicity

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Mercury (Hg) is one of the most toxic elements in the periodic table. Although Hg is present in nature, it has also been released into the environment for centuries as a result of anthropogenic activities. Nowadays, there are efforts to reduce its anthropogenic use; however, its environmental presence is significant and will persist. We are pleased to present this special issue on mercury toxicity. The objective of collecting research findings in a single issue devoted to the toxicology of mercury was to compile reports on the latest findings on Hg's toxicity from renowned research groups across the world. This special issue affords the opportunity to bring together a wide range of review and research papers devoted both to basic and applied toxicity associated with various exposure scenarios and Hg species (dental material, iatrogenic ethylmercury, fish-methylmercury) along with comprehensive description on experimental models. While human studies demonstrated the noxious effects of these forms of Hg, experimental studies have assisted in defining mechanistic pathways central to Hg's toxicity in various tissues and organ systems.

The volume is dedicated in part to articles that provide new insights on important considerations of subtle effects of exposure to multiple forms of organic mercury (ethylmercury in thimerosal-containing vaccines and methylmercury (MeHg) derived from maternal fish consumption) and neurological outcomes in infants (J. G. Dórea et al.). In addition, hypersensitivity to low-dose Hg exposure from dental amalgam fillings is detailed, showing exquisite sensitivity to amalgam-derived Hg in sensitized individuals (H. McParland and S. Warnakulasuriya). Local effects of amalgam and Hg dental restoration represent the most important nonoccupational exposure to inorganic mercury, while fish consumption represents the most common source of MeHg exposure.

The impacts of exposure to fish-derived MeHg at levels below those considered to pose neurological risk (hair level: 50 μg/g) were explored by Japanese researchers in subjects of the Niigata mercury poisoning (K. Maruyama et al.). Experimental research papers from this issue confirmed and extended observations that exposure of immature rodents to different chemical forms of Hg is associated with differential bodily distribution Hg (M. Blanuša et al.; C.-F. Huang et al.). C.-F. Huang et al. demonstrated that exposure of developing rats to cinnabar (HgS) caused long-lasting neurobehavioral and neurochemical toxic effect, indicating that the use of this millenary component of traditional Chinese medicine continues to represent a toxicological concern. Using an important, yet little explored experimental mouse model, J. P. Bourdineaud and colleagues demonstrated that the ingestion of MeHg-adulterated fish led to higher neurotoxicity in comparison to the ingestion of the "free salt" of methylmercury chloride (MeHgCl). The scarcity of studies on this subject highlights the need for future studies to address these persistent toxicological issues.

The molecular, subcellular, cellular, and systemic toxicity of Hg was also addressed here in this volume. The cardiovascular toxicity of Hg in humans and rodents was reviewed by B. F. Azevedo et al. The impact of Hg exposure on endothelial cell physiology is well established; however, the limit of dietary-derived Hg needed to trigger cardiotoxic effects is still debatable. The negative impact of oral exposure to Hg(II) on reproductive performance of male rats was demonstrated by J. C. Heath and collaborators, highlighting the need for detailed studies to determine the nonobservable adverse effect level (NOAEL) of Hg(II) in the male reproductive system, as well as Hg deposition in target tissues. The comparative renal and hepatic toxicity of Hg(II) and MeHg in fish was addressed by V. Branco et al., demonstrating that both forms of mercury targeted the antioxidant selenoenzyme thioredoxin-reductase (TrxR) and reinforcing the central role of disrupted selenoprotein function in mercurial toxicity. The *in vitro* and *in vivo* targeting of the critical sulfhydryl-containing enzyme, Na^+, K^+-ATPase was reviewed by I. Kade and addressed by T. S. Huang et al., noting divergent effects *in vitro* and *in vivo*. The role of mitochondria and calcium in the neurotoxicity of MeHg was reviewed by D. Roos et al., providing evidence that Ca^{2+}, glutamate, oxidative stress, and mitochondria play a central role in its neurotoxicity. The efficacy of the marine *n*-3 fatty acids, eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) in attenuating MeHg-induced toxicity was studied in fish and mammalian cell cultures. O. J. Nøstbakken et al. demonstrated that DHA decreased MeHg uptake into mammalian cells but increased MeHg-induced apoptosis in fish cells.

We hope that the new findings on the subtle effects of combined exposure to iatrogenic ethylmercury (from thimerosal-containing vaccines) and maternal MeHg (from fish consumption), as well as the results of experimental studies and the critical reviews presented herein can shed novel information on mercury's absorption, distribution, metabolism, and excretion, as well as its ill effects at the cellular, molecular, and organismal levels. Understanding of these facets of research is required for derivation on environmental and health policies as well as guidance for the most promising future research venues. Finally, we would like to thank all the reviewers that have contributed their time and insight to this special issue as well as the journal's personnel (particularly Doaa Hassan) for their support and making possible the publication of this special issue.

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